

8320157D.TRN  
November 13, 1996

KEEP WITH FILE 08320157D.TRN

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FILE 'USPAT' ENTERED AT 07:10:44 ON 13 NOV 96

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\* WELCOME TO THE \*  
\* U.S. PATENT TEXT FILE \*  
\*\*\*\*\*

=> s (cdn? or cdi?) not (cdna?)  
4929 CDN?  
1259 CDI?  
4679 CDNA?  
L1 1479 (CDN? OR CDI?) NOT (CDNA?)

=> s apoptosis  
L2 71 APOPTOSIS

=> s l1 and l2  
L3 1 L1 AND L2

=> d l3 cit ab

1. 5,559,038, Sep. 24, 1996, Gas chromatography/mass spectrometry determination of oxidized sulfhydryl amino acids; J. Fred Kolhouse, et al., 436/86, 120, 161, 173, 174, 178 [IMAGE AVAILABLE]

US PAT NO: 5,559,038 [IMAGE AVAILABLE] L3: 1 of 1

ABSTRACT:

Method for quantifying the amounts of oxidized sulfhydryl amino acids, particularly cysteine sulfinic acid, cysteic acid, homocysteine sulfinic acid and homocysteic acid, for example, in biological samples are provided. The oxidized sulfhydryl amino acids are measured by gas chromatography/mass spectrometry after derivatization, preferably using stable isotope internal standards. Deviation from normal individuals' levels of oxidized sulfhydryl amino acids in serum are indicative of certain neuropsychiatric disorders, the biochemical origin of which can be diagnosed using methods disclosed herein, particularly when relative levels of these and/or other sulfur amino acids are measured.

=> quit  
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF  
LOGOFF? (Y)/N/HOLD:y

U.S. Patent & Trademark Office LOGOFF AT 07:11:40 ON 13 NOV 96

8320157B.TRN  
November 13, 1996

KEEP WITH FILE 08320157B.TRN

FILE 'HOME' ENTERED AT 07:02:40 ON 13 NOV 96

=> file .lit1

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		0.15	0.15

FILE 'MEDLINE' ENTERED AT 07:03:21 ON 13 NOV 96

FILE 'BIOSIS' ENTERED AT 07:03:21 ON 13 NOV 96

COPYRIGHT (C) 1996 BIOSIS(R)

FILE 'EMBASE' ENTERED AT 07:03:21 ON 13 NOV 96

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FILE 'CAPLUS' ENTERED AT 07:03:21 ON 13 NOV 96

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(FILE 'HOME' ENTERED AT 07:02:40 ON 13 NOV 96)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 07:03:21 ON 13  
NOV 96

L1 4448 FILE MEDLINE

L2 4598 FILE BIOSIS

L3 4145 FILE EMBASE

L4 7135 FILE CAPLUS

TOTAL FOR ALL FILES

L5 20326 S (CDN? OR CDI?) NOT (CDNA)

L6 7541 FILE MEDLINE

L7 12113 FILE BIOSIS

L8 8406 FILE EMBASE

L9 7502 FILE CAPLUS

TOTAL FOR ALL FILES

L10 35562 S APOPTOSIS

L11 15 FILE MEDLINE

L12 18 FILE BIOSIS

L13 17 FILE EMBASE

L14 18 FILE CAPLUS

TOTAL FOR ALL FILES

L15 68 S L5(P)L10

L16 446367 FILE MEDLINE

L17 373775 FILE BIOSIS

L18 327137 FILE EMBASE

L19 216157 FILE CAPLUS

TOTAL FOR ALL FILES

L20 1363436 S ANTIBOD?

L21 29 DUP REM L15 (39 DUPLICATES REMOVED)

=> s l15 not (cdnas)

L22 1 FILE MEDLINE

L23 2 FILE BIOSIS

L24 1 FILE EMBASE

L25 1 FILE CAPLUS

TOTAL FOR ALL FILES

L26 5 L15 NOT (CDNAS)

=> dup rem l26

PROCESSING COMPLETED FOR L26

L27 3 DUP REM L26 (2 DUPLICATES REMOVED)

=> d l27 1- bib ab

L27 ANSWER 1 OF 3 BIOSIS COPYRIGHT 1996 BIOSIS

AN 96:191087 BIOSIS

DN 98747216

TI Differentiation of U-937 promonocytic cells with mitomycin C or cis-diamminedichloroplatinum II.

AU Ballester A; Perez C; Aller P; Mata F

CS Dep. Bioquímica Biología Molecular I, Fac. Biol., Univ. Complutense, 28040 Madrid, Spain

SO International Journal of Cancer 65 (6). 1996. 791-795. ISSN: 0020-7136

LA English

AB Administration of 0.3  $\mu$ M mitomycin C (MMC) or 2.0  $\mu$ M cis-diamminedichloroplatinum II (CDDP) decreased the growth activity and induced the differentiation of U-937 human promonocytic cells, as shown by nitroblue tetrazolium reduction and an increase in surface expression of the leukocyte integrins **\*\*\*CD11b\*\*\*** /CD18 and **\*\*\*CD11c\*\*\*** /CD18. Expression of these differentiation markers started to be significant at 48 hr of treatment. These concentrations resulted in little cell damage (determined by Trypan blue exclusion) and slightly induced **\*\*\*apoptosis\*\*\*** (determined by DNA degradation and changes in nuclear morphology). The treatments induced a transient increase in c-fos and c-jun mRNA levels, with maximum values at 1-6 hr; a transient increase in collagenase mRNA level, with a maximum value at 48 hr; and a progressive increase in vimentin and lamin A and C mRNA levels. These changes were qualitatively similar to those produced by 12-O-tetradecanoylphorbol 13-acetate. CDDP and MMC also caused a transient increase of total AP-1 binding activity, as determined by gel retardation assays. The drugs produced an early transient activation (3-6 hr) of membrane-bound protein kinase C, followed by a later activation (48 hr) of both the membrane and the cytosolic enzyme. These results suggest that protein kinase C and AP-1-dependent gene expression could be involved in myeloid cell differentiation by alkylating agents.

L27 ANSWER 2 OF 3 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 1

AN 96:311792 BIOSIS

DN 99034148

TI The dual role of S-nitrosoglutathione (GSNO) during thymocyte **\*\*\*apoptosis\*\*\***.

AU Sandau K; Bruene B

CS Univ. Erlangen-Nuernberg, Fac. Med., Dep. Med. IV-Experimental Div., 91054 Erlangen, Germany

SO Cellular Signalling 8 (3). 1996. 173-177. ISSN: 0898-6568

LA English

AB Nitric oxide (NO) is known to regulate redox-sensitive signalling

pathways in physiology and pathophysiology. Depending on its concentration, the NO-releasing compound S-nitrosoglutathione (GSNO) causes negative and positive regulation of thymocyte

\*\*\*apoptosis\*\*\*. At levels below 0.6 mM, GSNO produces deoxyribonucleic acid (DNA) laddering, which is inhibited by activation of protein kinase C (PKC), cycloheximide treatment, and calcium chelation. Higher concentrations of the NO donor (1-2 mM) suppress thymocyte \*\*\*apoptosis\*\*\* initiated by the classical agonist dexamethasone. Inhibition of \*\*\*apoptosis\*\*\* by NO is analogous to the action of the thiol-blocking compound N-ethylmaleimide (NEM) and the glutathione-S-transferase substrate 1-chloro-2,4-dinitrobenzene (\*\*\*CDNB\*\*\*). Inhibition of \*\*\*apoptosis\*\*\* results from thiol modification of critical proteins in response to NO treatment. Depending on the concentration, GSNO can be involved either in toxic or in protective signalling in thymocyte biology.

L27 ANSWER 3 OF 3 MEDLINE

AN 95370107 MEDLINE

TI Bcl-2: prevention of \*\*\*apoptosis\*\*\* as a mechanism of drug resistance.

AU Reed J C

CS La Jolla Cancer Research Foundation, Cancer Research Center, California, USA..

SO HEMATOLOGY/ONCOLOGY CLINICS OF NORTH AMERICA, (1995 Apr) 9 (2) 451-73. Ref: 89

Journal code: HEO. ISSN: 0889-8588.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, ACADEMIC)

LA English

FS Priority Journals

EM 9511

AB Programmed cell death (also known as \*\*\*apoptosis\*\*\* ) plays an essential role in tissue homeostasis, where it ensures that new cell production in the body is offset by a commensurate rate of cell loss. Defects in the genetic pathway that regulate the cell death process can figure prominently in the origins of cancer and also in problems with cancer treatment. Eventually, it may be possible to develop novel treatments for cancer that specifically seek to modulate the physiologic cell death pathway as opposed to nearly all currently available drugs, which are intended to interfere with some aspect of the cell division cycle.

=> file wpids

COST IN U.S. DOLLARS		SINCE FILE	TOTAL
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ENTRY	SESSION
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FULL ESTIMATED COST	35.41	35.56
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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE	TOTAL
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ENTRY	SESSION
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CA SUBSCRIBER PRICE	-1.85	-1.85
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FILE 'WPIDS' ENTERED AT 07:08:56 ON 13 NOV 96

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FILE LAST UPDATED: 11 NOV 96 <961111/UP>  
 >>>UPDATE WEEKS:  
 MOST RECENT DERWENT WEEK 9645 <199645/DW>  
 DERWENT WEEK FOR CHEMICAL CODING: 9634  
 DERWENT WEEK FOR POLYMER INDEXING: 9642  
 DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE  
 >>> DERWENT POLYMER INDEXING THESAURUS AVAILABLE IN FIELD /PLE <<<  
 >>> PATENT IMAGES AVAILABLE FOR PRINT AND DISPLAY <<<  
 >>> PATENTS CITATION INDEX AVAILABLE AS FILE DPCI <<<

=> s (cdn? or cdi?) not (cdna?)  
 2241 CDN?  
 190 CDI?  
 2206 CDNA?  
 L28 225 (CDN? OR CDI?) NOT (CDNA?)

=> s apoptosis  
 L29 141 APOPTOSIS

=> s l28 and l29  
 L30 1 L28 AND L29

=> d l30 1- bib ab

L30 ANSWER 1 OF 1 WPIDS COPYRIGHT 1996 DERWENT INFORMATION LTD  
 AN 95-215106 [28] WPIDS  
 DNC C95-099466

TI New nucleic acid sequences encoding \*\*\*Cdn\*\*\* \*\*\*apoptosis\*\*\*  
 modulators - and related vectors, transformed cells, proteins and  
 antibodies, useful for diagnosis and treatment e.g. of HIV  
 infection, reperfusion injury etc..

DC B04 D16

IN BARR, P J; KIEFER, M C

PA (LXRB-N) LXR BIOTECHNOLOGY INC

CYC 20

PI WO 9515084 A1 950608 (9528)\* EN 66 pp

AU 9513351 A 950619 (9540)

NO 9602188 A 960723 (9639)

EP 731636 A1 960918 (9642) EN

R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE

FI 9602247 A 960722 (9642)

ADT WO 9515084 A1 WO 94-US13930 941130; AU 9513351 A AU 95-13351 941130;

NO 9602188 A WO 94-US13930 941130, NO 96-2188 960529; EP 731636 A1

WO 94-US13930 941130, EP 95-904808 941130; FI 9602247 A WO

94-US13930 941130, FI 96-2247 960529

FDT AU 9513351 A Based on WO 9515084; EP 731636 A1 Based on WO 9515084

PRAI US 94-320157 941007; US 93-160067 931130

AB WO 9515084 A UPAB: 950721

Purified nucleic acid (I) encoding a \*\*\*Cdn\*\*\* is new. Also new  
 is pure natural or recombinant \*\*\*Cdn\*\*\* protein.

USE - \*\*\*Cdn\*\*\* proteins are modulators of

\*\*\*apoptosis\*\*\* which can be controlled by altering endogenous

\*\*\*Cdn\*\*\* levels, either by increasing expression of a recombinant

or endogenous gene or by admin. of \*\*\*Cdn\*\*\* protein.

\*\*\*Cdn\*\*\* can be used in all cases where superoxide dismutase is a  
 suitable treatment e.g. HIV infection, autoimmune disease, cardiac  
 or neuronal diseases, hepatitis, osteoporosis, shock or

proliferative disease (e.g. B cell lymphoma or eczema). Some partic. applications are ex vivo transfection of T cells with (I) before return to an HIV-positive subject to increase T cell survival time, in vivo transfection to prevent atherosclerosis, prevention of reperfusion injury in cerebral or myocardial infarct, as a pretreatment before admin. of cardiotoxic drugs, etc. Ab can be used to detect \*\*\*Cdn\*\*\* expression for diagnosis or monitoring; abnormal levels of \*\*\*Cdn\*\*\* are likely to indicate inappropriate \*\*\*apoptosis\*\*\*.

ADVANTAGE - Control of \*\*\*Cdn\*\*\* expression allows very specific modulation of \*\*\*apoptosis\*\*\*.

Dwg.0/11

=> d his

(FILE 'HOME' ENTERED AT 07:02:40 ON 13 NOV 96)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 07:03:21 ON 13 NOV 96

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L1      4448 FILE MEDLINE
L2      4598 FILE BIOSIS
L3      4145 FILE EMBASE
L4      7135 FILE CAPLUS
TOTAL FOR ALL FILES
L5      20326 S (CDN? OR CDI?) NOT (CDNA)
L6      7541 FILE MEDLINE
L7      12113 FILE BIOSIS
L8      8406 FILE EMBASE
L9      7502 FILE CAPLUS
TOTAL FOR ALL FILES
L10     35562 S APOPTOSIS
L11     15 FILE MEDLINE
L12     18 FILE BIOSIS
L13     17 FILE EMBASE
L14     18 FILE CAPLUS
TOTAL FOR ALL FILES
L15     68 S L5(P)L10
L16     446367 FILE MEDLINE
L17     373775 FILE BIOSIS
L18     327137 FILE EMBASE
L19     216157 FILE CAPLUS
TOTAL FOR ALL FILES
L20     1363436 S ANTIBOD?
L21     29 DUP REM L15 (39 DUPLICATES REMOVED)
L22     1 FILE MEDLINE
L23     2 FILE BIOSIS
L24     1 FILE EMBASE
L25     1 FILE CAPLUS
TOTAL FOR ALL FILES
L26     5 S L15 NOT (CDNAS)
L27     3 DUP REM L26 (2 DUPLICATES REMOVED)

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FILE 'WPIDS' ENTERED AT 07:08:56 ON 13 NOV 96

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L28     225 S (CDN? OR CDI?) NOT (CDNA?)
L29     141 S APOPTOSIS
L30     1 S L28 AND L29

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=> quit h

COST IN U.S. DOLLARS

ENTRY	SINCE FILE	SESSION	TOTAL
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FULL ESTIMATED COST	3.53		39.09
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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)

ENTRY	SESSION
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CA SUBSCRIBER PRICE	0.00	-1.85
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SESSION WILL BE HELD FOR 60 MINUTES